

Suspected calcium oxalate raphide irritation in a black rhinoceros (*Diceros bicornis*) due to ingestion of *Xanthosoma mafaffa*

We wish to record a case of buccal irritation in a black rhinoceros, 'Chewore', due to chewing of *Xanthosoma mafaffa* at the Tashinga Camp, Matusadona National Park, Kariba, Zimbabwe, on 27 April 1995. The rhinoceros was approximately 12 months old and weighed 385 kg. The incident occurred when she ate an ornamental elephant-ear plant from a garden. About half a leaf was chewed and spat out; nothing was swallowed. The time from intake to onset of symptoms, manifested as a flow of white, foamy saliva, was 10–15 minutes. This stopped 15 minutes after 1 l of milk had been given. Five minutes after the salivation had ceased she started flicking her tongue in and out of her mouth as if she was trying to rub something off. This lasted approximately half an hour. She then went to a mud wallow and after first using her horn to dig at the edge, she rubbed her mouth in the mud for about 20 minutes. She was hosed down with water at the same time. After that, she lay down for an hour and then went for a walk. She tried to browse but food was only chewed and spat out, not swallowed. Three hours after chewing the plant she returned from her walk and was fed 3 l milk and then ate 350 g of rhinoceros cubes mixed with a little molasses, after which she started browsing and swallowing. Seven hours after chewing the plant she took another 35 l of milk and by this time appeared normal. She did not groan at all, and apart from being obviously distressed during the incident, showed no other overt signs of illness. The symptoms lasted 3–4 hours and no special treatment was administered.

The causative plant was identified by the National Herbarium of Zimbabwe as *Xanthosoma mafaffa* Schott (family Araceae). According to Purseglove⁷, *Xanthosoma* (new cocoyam, tannia, yautia) has

its origin in the northern parts of South America. *X. mafaffa*, in addition to being an ornamental garden plant, is widely cultivated, particularly in West Africa, on the same scale as *Colocasia esculenta* (true cocoyam, dasheen, madumbe, taro), for its starch-rich tuberous rhizomes. The green leaves are also used as a vegetable². It is, however, essential to boil or bake these plants before eating to destroy raphides that have a severely acrid effect on the buccal mucous membrane⁷.

Members of the Araceae are well-known to contain such calcium oxalate raphides (needle-sharp, grooved crystals up to 250 µ long) that are packed tightly in parallel in special ejector cells in the plant tissues⁴. When mechanical damage occurs, for example when the plant is chewed, the ejector cells explode, releasing masses of the irritating crystals into the mouth. The resultant severe but fortunately transient irritation is ascribed to both the mechanical damage caused by the sharp crystals and penetration of the damaged mucous membrane by the high concentration of free oxalic acid and possibly other irritants contained in these plants³. Raphides and the associated toxins may have an antiherbivory function⁸.

The incident is typical of this intoxication, which occurs quite often in man and has been encountered in this region in puppies and small stock exposed to common araceous garden plants such as *Zantedeschia* and *Colocasia* spp. but which, owing to its transient and non-fatal nature, is not reported. The latent period is some 30 seconds in man (pers. obs., TWN) and this fits in with the history that the leaf was chewed but soon spat out.

The black rhinoceros is known to browse extensively on several of the highly irritant, latex-containing *Euphorbia* spp. such as *E. virosa* in Namibia⁶ and *E. ingens* and *E. grandicornuta* in KwaZulu-

Natal, South Africa (G Hughes and D V Cooper, Natal Parks Board, pers. comm. 1996) without any apparent damage to the buccal mucous membrane. Domestic ruminants⁵ and man⁹, in contrast, are severely affected if they attempt to eat these plants or even have skin contact with the latex. It is, therefore, extraordinary that a relatively much less irritating plant like *X. mafaffa* would have such a clearly irritating effect in this species.

This occurrence again emphasizes the danger of exposing naive game to common garden plants, sometimes with fatal results¹.

We thank Prof. D G Meltzer, Faculty of Veterinary Science, University of Pretoria, Onderstepoort, for facilitating contact between the authors.

REFERENCES

1. Brain C, Fox V E B 1994 Suspected cardiac glycoside poisoning in elephants (*Loxodonta africana*). *Journal of the South African Veterinary Association* 65: 173–174
2. Burkill H M 1985 *The useful plants of west tropical Africa* Vol. 1 (2nd edn). Royal Botanical Gardens, Kew
3. Frohne D, Pfänder H J 1984 *A colour atlas of poisonous plants*. Wolfe Publishing, London
4. Genua J M, Hillston C J 1985 The occurrence, type and location of calcium oxalate crystals in the leaves of fourteen species of Araceae. *Annals of Botany* 56: 351–361
5. Kellerman T S, Coetzer J A W, Naudé T W 1988 *Plant poisonings and mycotoxicoses of livestock in southern Africa*. Oxford University Press, Cape Town
6. Loutit B D, Louw G N, Seely M K 1987 First approximation of food preferences and the chemical composition of the diet of the desert-dwelling black rhinoceros, *Diceros bicornis* L. *Madoqua* 15: 35–54
7. Purseglove J W 1972 *Tropical crops: monocotyledons*. Longman Group, London
8. Singh Y 1996 Contributions to the systematics of the genus *Zantedeschia* Spreng. (Araceae). MSc thesis, University of Pretoria
9. Watt J M, Breyer-Brandwijk M G 1962 *The medicinal and poisonous plants of southern and eastern Africa*. E & S Livingstone, Edinburgh

P A Wood^a, D C Foggin^b and T W Naudé^c

^aMatusadona National Park, Private Bag 2003, Kariba, Zimbabwe.

^bDepartment of Veterinary Services, PO Box CY 66, Causeway, Zimbabwe.

^cDivision of Toxicology, Onderstepoort Veterinary Institute, Private Bag X05, Onderstepoort, 0110 South Africa.

Suspected aflatoxicosis in breeding budgerigars

Towards the end of December 1995/ beginning of January 1996 mortalities occurred in a budgerigar breeding colony

in the Krugersdorp district of the Gauteng Province in South Africa. The flock consisted of ca. 100 birds, including 10

breeding pairs with 18 nestlings. Mortalities were limited to the breeding colony and only adult birds were affected. Four

breeding birds which had recently been purchased from another breeder were not affected and no mortalities were recorded amongst the fledgeling birds which are kept in the cage adjacent to that of the breeding colony.

The affected birds developed a watery, yellow-green diarrhoea which persisted for several weeks. Treatment with a coccidiostatic preparation (ESP³, Ciba, Janssen AH), and a tetracycline antibiotic/multivitamin preparation (Doxybiotic, Medpet) did not clear the diarrhoea and the birds eventually died. At the time that the necropsies were performed, a large proportion of the breeding flock had died.

Three birds that died were submitted to the Pathology Section of the Onderstepoort Veterinary Institute (OVI) for necropsy.

The carcass of the first bird (Case 1) had been opened before submission to OVI. As the organs were contaminated with feathers and debris, no specimens were taken for bacterial isolation and the owner was advised to submit additional unopened carcasses for necropsy. This bird revealed diffuse moderate congestion and the gizzard contained only a small amount of seeds.

A week later another 2 birds were received for necropsy (cases 2 and 3). The carcasses of these birds were severely congested and dehydrated with signs of a watery, greenish diarrhoea at the vent. The livers of both birds were moderately enlarged and the fat depots appeared to be diminished. In case 2 the liver was normal in colour but in case 3 it was bright yellow and friable with scattered multifocal 1–2 mm white foci. The spleen of both birds was enlarged, particularly in case 3 and the kidneys were swollen, friable and pale brown. The gizzard of case 2 contained a small amount of seeds and a few stones 1–2 mm in diameter, while that of case 3 was empty. The intestines of both birds contained watery, brown fluid ingesta.

On histopathological examination, the livers of cases 1 and 2 revealed moderate diffuse fatty change of the hepatocytes (Fig. 1), the cytoplasm of which contained numerous small vacuoles (foamy appearance) or a single to a few large vacuoles. The hepatocyte nuclei were moderately enlarged and vesicular and scattered cells revealed anisonucleosis or occasionally binucleation. A few eosinophilic, rounded cytosegrosomes were present loose in the sinusoids or within the cytoplasm of the hepatocytes. The portal tracts revealed severe and extensive mixed cellular infiltration (Fig. 1) of lymphocytes, plasma cells, macrophages (some of

which contained golden-brown pigment granules) and scattered heterophils. Indistinct, proliferating bile ducts were evident at the edges of the cellular infiltrates (Fig. 1). The hepatocytic cytoplasmic and nuclear changes in case 3 were similar to those of cases 1 and 2 but the fatty changes were more pronounced. The hepatocytes in this bird were mildly enlarged and aberrant in shape. The portal inflammatory reaction was similar to that of cases 1 and 2 but more pronounced and more chronic with proportionately more pigmented macrophages and fewer heterophils. Numerous prominent and distinct, proliferating bile ductules were present at the edges of and amongst the portal inflammatory cells and intralobularly bridging across adjacent lobules. Several cytosegrosomes and shrunken, necrotic hepatocytes were present amongst the cells in the portal tracts as well as accumulations of light to bright pink, eosinophilic material, probably proteinaceous fluid. Scattered cytosegrosomes were present in the sinusoids or within hepatocytes as described for cases 1 and 2 and there was also mild single-cell necrosis of the hepatocytes.

Lesions in other tissues included a mild degenerative nephrosis of the cortical tubules and multifocal interstitial lymphoid nephritis; splenic lymphoid depletion, haemosiderosis and plasmacytosis; serous atrophy of the mesenteric fat; and, pulmonary congestion and oedema.

Specimens of liver, spleen, lung and small intestine from cases 2 and 3 were submitted for isolation of aerobic bacteria

and fungi. No pathogenic bacteria were isolated but *Aspergillus flavus* was cultured from the liver and lung of case 2 and the liver and intestine of case 3, the highest concentration being in the liver of case 2.

Impression smears of the liver, lung and spleen from cases 2 and 3 were negative for *Chlamydia psittaci* via the immunofluorescent antibody technique.

A diagnosis of suspected aflatoxicosis was made based on the histopathological findings of fatty change and bile ductule proliferation in the livers, and the isolation of *A. flavus* from samples of liver, gut and lung from cases 2 and 3. Confirmation of aflatoxicosis, however, rests on the finding of toxic levels of aflatoxins in the feed. This was not done in this outbreak owing to financial constraints, as analysis of feed for aflatoxins is expensive.

Gross necropsy lesions in birds suffering from aflatoxicosis can include an enlarged, pale fatty liver, splenomegaly, an enlarged pancreas, atrophy of the cloacal bursa and diminished fat depots², several of which were evident in the birds necropsied in this outbreak. The histopathological features of the hepatic lesions, in particular fatty change of hepatocytes and bile ductule proliferation in all 3 cases, are highly suggestive of aflatoxicosis^{1,2}. Of interest was the isolation of *A. flavus*, particularly from the gut and liver, as this indicates ingestion of this fungus by the affected birds and points to the presence and possible growth of this fungus on the feed.

The pyogranulomatous inflammation of the portal tracts in all 3 birds can probably be attributed to a secondary

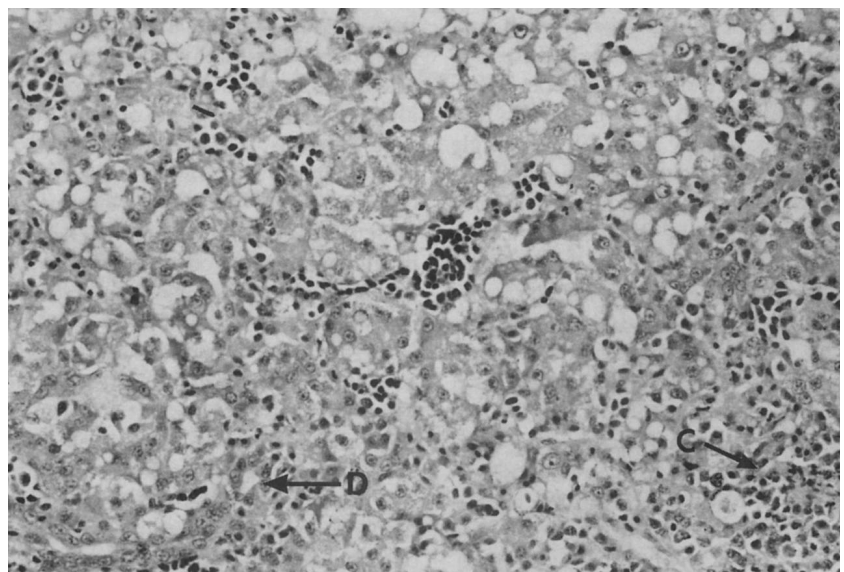


Fig. 1: Liver, Case 2: diffuse hepatocytic fatty change; groups of indistinct proliferating bile ductules (arrow D); portal cellular infiltrates (arrow C). HE, $\times 110$.

bacterial infection of a chronic nature. Aflatoxicosis is known to cause immunosuppression by inhibiting protein and nucleic acid synthesis, which could have predisposed the affected birds to bacterial infection².

The 4 principal mycotoxins of concern in birds are aflatoxin B₁, ochratoxin A, deoxynivalenol (vomitoxin) and the trichothecenes. The moulds producing these toxins can grow on various feeds, including grains, peanuts, breads, meat and cheese². The birds in this outbreak were fed seeds but the owner would not allow the feed to be examined for mould

and it could not be determined whether all the birds received the same ration. As only the breeding birds were affected it is possible that this group was offered supplements not supplied to the other groups. Breeding birds would also have higher nutritional needs and could hence consume higher, possibly toxic levels of toxins present in or on the feed than birds in other groups.

The isolation of *A. flavus* from the liver and gut of the affected birds lends support to a diagnosis of aflatoxicosis. In cases/outbreaks where it is not possible or economically feasible to analyse the feed

for aflatoxin mycotoxins, consideration can be given to using standard methods to isolate fungi from the liver and gut. If *A. flavus* is found this can lend support to the histopathological findings in making a diagnosis of aflatoxicosis.

REFERENCES

1. Kelly R F 1992. The liver and biliary system. In Jubb K V F, Kennedy P C, Palmer N (eds) *Pathology of domestic animals* Vol. 2 (4th edn). Academic Press, San Diego: 319–406
2. Dumonceaux G, Harrison G J 1994. Toxins. In Ritchie B W, Harrison G J, Harrison L R (eds) *Avian medicine*. Wingers Publishing, Lake Worth: 1034–1049

Stella S Bastianello^a, C C Dolman^c, Marijke M Henton^b and Mary-Louise Penrith^a

^aPathology Section, Onderstepoort Veterinary Institute, Onderstepoort, 0110 South Africa.

^bBacteriology Section, Onderstepoort Veterinary Institute, Onderstepoort, 0110 South Africa.

^cCorlett Small Animal Clinic, PO Box 3098, Kenmare, 1745 South Africa.

The possible role of manganese poisoning in enzootic geophagia and hepatitis of calves and lambs

Geophagia, or the deliberate ingestion of soil, has been classified as a form of pica, which is usually defined as the compulsive ingestion of inappropriate or foreign material^{1,5,8,9}. A specific enzootic form of geophagia occurs in young cattle and sheep on manganese-rich soil derived from the weathering of superficial dolomitic (carbonate) rock formations in the Northern Cape and North-West Provinces. It results in severe subacute to chronic cholangiohepatitis, icterus and a high mortality rate in untreated cases.

Organ specimens from calves characterised by marked icterus, large amounts of ingested black to dark-brown soil, and a severe chronic cholangiohepatitis, have been referred to the Onderstepoort Veterinary Institute (OVI) by veterinarians and farmers sporadically since 1972. A single lamb submitted had similar lesions. These cases were at first difficult to interpret and were suspected to be due to a plant toxicosis or a chronic infection of the liver. The condition was given the vernacular name of 'Vryburg hepatosis', derived from the district in the North-West Province whence most of the cases originated and the characteristic histopathological changes observed in the liver.

The problem apparently only occurs on 14 farms in the Vryburg, Postmasburg and Barkly West districts of the Northern Cape and North-West Provinces of South Africa. These farms all have outcrops of dolomitic rock reported to be rich in manganese^{2,3}. An association between the disease and

manganese was therefore suspected.

The soil on the affected farms contains numerous small, round to ovoid, black-grey manganese-rich iron-containing concretions *ca.* 1–10 mm in diameter. Young calves display an insatiable appetite for the manganese-rich soil, sometimes also licking iron poles. The highest frequency of geophagia occurs at 7–14 days, calves older than about 2 months of age rarely being affected. No specific sex or breed predilection could be determined. The symptoms reported usually started with intermittent, progressively worsening geophagia, followed by constipation, dehydration and death within about 7–10 days in untreated cases. Macroscopical pathological changes in affected calves consisted of moderate to severe icterus, a markedly enlarged and yellow liver, and variable amounts of dark-brown to black soil, largely composed of small black to dark-brown manganese-rich concretions, in the stomach and intestines. Histopathology revealed marked cholangiohepatitis of some duration. Morbidity and mortality rates were difficult to estimate accurately, since farmers applied certain preventative measures described below, but owners on severely affected farms estimated that about 50–75 % of calves may develop geophagia, with a mortality rate of close to 100 % in untreated cases.

Liver specimens from 23 calves and 1 lamb with soil in their digestive tracts or a history of geophagia were examined by

Atomic Absorption Spectrometer and revealed high concentrations of manganese, ranging from 10–1800 ppm wet mass (WM) (normal range 2–3 ppm WM)^{4,6}. Since liver specimens from 4 full-term bovine foetuses that had died during dystocia, 4 neonatal calves that had not yet ingested milk, 11 yearling tollies and 15 culled cows from 2 severely affected farms had manganese and iron content within normal limits, it appeared that high levels of manganese must be taken in and accumulated during the pre-weaning period after the ingestion of manganese-rich soil or milk. It was necessary to establish not only the source of the manganese, but whether the observed effects were directly due to manganese intoxication or were the result of mineral interactions, for example suppression of absorption of elements such as iron and cobalt, by manganese.

Milk specimens from cows with calves 1–12 weeks of age from 3 severely affected farms had higher levels of manganese and lower levels of iron than cows under similar grazing conditions at the Onderstepoort and Irene experimental farms chosen as control levels for reference. Grazing plants (red grass, *Themeda triandra*, and rosyntjebos, *Grewia flava*) from affected farms collected during summer 1992 and autumn 1995 revealed higher levels of manganese, and a higher ratio of manganese to iron, than plants from unaffected farms near Vryburg, Irene and Onderstepoort (H J C Smith,